

Photobiomodulation at 660nm Stimulates Fibroblast Differentiation

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Background and Objectives: Among many of the different complications that diabetic patients suffer, foot ulcers are the most challenging, and in many cases result in non-traumatic lower limb amputation and permanent disability.

To alleviate this burden, new interventions such as photobiomodulation (PBM) have been utilized. However, the cellular pathways affected by PBM have not yet been fully recognized. The differentiation of fibroblasts into myofibroblasts forms a vital part of wound healing and is often impaired under diabetic conditions. Therefore, this study sought to investigate the effects of PBM at 660nm on the transforming growth factor- β 1 (TGF- β 1)/Smad pathway and the differentiation of fibroblasts into myofibroblasts.

Study Design/Materials and Methods: WS1 fibroblasts were treated with PBM using a wavelength of 660 nm at a fluence of 5 J/cm² in normal, normal wounded, diabetic, and diabetic wounded models. Post-irradiation cellular responses were observed at 24, 48, and 72 hours to ascertain morphological changes and cell viability, and the expression of fibroblast differentiation markers (Thy-1 or CD90, extra domain A fibronectin or EDA-FN and α -smooth muscle actin or α -SMA), TGF- β 1, phosphorylated (p)TGF- β receptor 1 (R1), and p-Smad2/3.

Results: There was a significant increase in cell viability in all irradiated cell models, and no real significant changes in TGF- β 1, pTGF- β 1R1, and p-Smad2/3. As incubation time post-irradiation increased, Thy-1 (CD90) decreased, while EDA-FN and α -SMA increased in wounded models.

Conclusions: PBM at 660 nm with 5 J/cm² was successful in stimulating the differentiation of fibroblasts into myofibroblasts in diabetic wounded cells, which was independent of the TGF- β 1/Smad pathway. Fibroblast transition into myofibroblasts is vital to wound healing, failure of which results in impaired healing; PBM is able to foster such a transition. *Lasers Surg. Med.* © 2019 Wiley Periodicals, Inc.